REPORT DOCUMENTATION PAGE

Form Approved
OMB No. 0704-0188

PLEASE DO NOT RETURN YOUR FORM TO THE ABOVE ORGANIZATION.

1. REPORT DATE *(DD-MM-YYYY)*	2. REPORT TYPE	3. DATES COVERED *(From - To)*
10/20/2017	Poster	10/20/2017-10/24/2017

4. TITLE AND SUBTITLE
Emergent craniotomy for SAH with cerebral-induced cardiac ischemia

5a. CONTRACT NUMBER

5b. GRANT NUMBER

5c. PROGRAM ELEMENT NUMBER

6. AUTHOR(S)
Capt Lee A Babbel

5d. PROJECT NUMBER

5e. TASK NUMBER

5f. WORK UNIT NUMBER

7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)
59th Clinical Research Division
1100 Willford Hall Loop, Bldg 4430
JBSA-Lackland, TX 78236-9908
210-292-7141

8. PERFORMING ORGANIZATION REPORT NUMBER
17409

9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES)
59th Clinical Research Division
1100 Willford Hall Loop, Bldg 4430
JBSA-Lackland, TX 78236-9908
210-292-7141

10. SPONSOR/MONITOR'S ACRONYM(S)

11. SPONSOR/MONITOR'S REPORT NUMBER(S)

12. DISTRIBUTION/AVAILABILITY STATEMENT
Approved for public release. Distribution is unlimited.

13. SUPPLEMENTARY NOTES
American Society of Anesthesiologists Annual Meeting 2017 (ASA), Boston MA 20-24 Oct 2017

14. ABSTRACT

15. SUBJECT TERMS

16. SECURITY CLASSIFICATION OF:			17. LIMITATION OF ABSTRACT	18. NUMBER OF PAGES	19a. NAME OF RESPONSIBLE PERSON
a. REPORT	b. ABSTRACT	c. THIS PAGE			Claricel Longoria
					19b. TELEPHONE NUMBER *(Include area code)* 210-292-7141

Approved for public release. Distribution is unlimited.

Emergent craniotomy for SAH with cerebral-induced cardiac ischemia

Babbel, Lee DO, Rosas, Alex MD

San Antonio Uniformed Services Health Education Consortium Anesthesiology Program

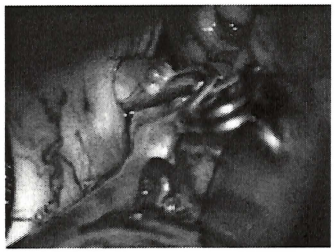
Case Presentation

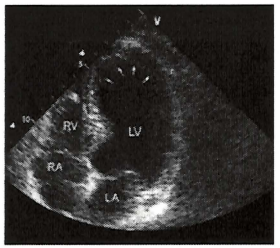
55 year old previously healthy female with a large SAH (HH 4) presented emergently to the OR for aneurysmal clipping for three cerebral aneurysms (anterior communicating artery , anterior cerebral, and basilar tip). In the ICU she had no hemodynamic instability but her EKG had changed from NSR with no T wave abnormalities to NSR with T wave inversions. Cardiac evaluation showed troponin elevation and peaked at 6.5.

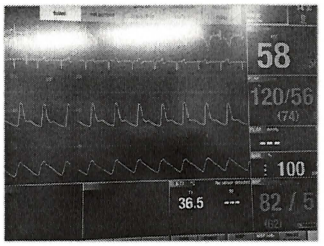
TTE showed LVEF 35-39% and an akinetic RV and LV apex and akinetic LV mid anterior and septal walls; thought to be secondary to Takotsubo cardiomyopathy.

In the OR 0.5 MAC Isoflurane and a propofol, precedex and hypertonic saline infusion were utilized . Cranial pinning was done with boluses of propofol and fentanyl to ensure normotension. Using the patients EVD CPP was maintained at approximately 60mmHg. Mannitol 75g was given at incision and ventilation was targeted to a PaCo2 of ~ 30mmHg prior to dural opening. Close attention to the patient's hemodynamics and carefully titrated norepinephrine and propofol boluses ensured hemodynamically stability throughout the case. Paralysis was maintained with vecuronium and no neuro monitoring was used. The three aneurysms were clipped and the neurosurgeon requested normotension throughout. She remained intubated post operatively.

Transcranial Doppler the next day showed moderate to severe left MCA vasospasm. Troponins continued to trend down and T wave inversions improved throughout the next several days but her neuro exam remained unchanged. She continued to have poor cardiac function and required prolonged use of norepinephrine and milrinone infusions for blood pressure augmentation.







Discussion

Following a SAH, injury to the posterior hypothalamus stimulates catecholamine release from the adrenal medulla and sympathetic cardiac afferents.

Norepinephrine, either through direct toxicity or by significant afterload elevation can produce ischemia in the sub endocardium. This may produce changes to the patient ECG as well as their cardiac function. These ischemic ECG and functional changes are not typically explained by underlying coronary disease or thrombosis.

Abnormalities in ECG tracings of rhythm and morphology are seen in 50-80% of patients with SAH. They usually appear within 48 hours and normalize in 6 weeks. The most common ECG changes are repolarization abnormalities and changes of in the T wave morphology and ST segment. The large upright T waves "cerebral T waves" are well described in patients with SAH. Ischemic changes such as ST and T wave changes correlate with the male gender and worse one year functional outcome.

Prolonged QTc is frequently seen and has been shown to correlate with the female gender and use of propofol. Cardiac dysrhythmias may also occur and can lead to life threatening dysrhythmias usually during the first 48 hours after SAH.

Troponin elevation is also frequently seen and is related to the severity of the intracranial hemorrhage. Patients with elevated troponin I concentrations are more likely to have ECG abnormalities and clinical evidence of left ventricular dysfunction.

The sub endocardial injury can manifest itself as a stress cardiomyopathy with reduced global LV systolic function and appear to have the Takotsubo cardiomyopathy appearance. This ventricular dysfunction is seen in up to 33% of patients after SAH is associated with pulmonary edema, intra atrial thrombus and embolic stroke.

Anesthetic management

Care of the patient with cerebral induced cardiac ischemia starts when possible, with pre-operative evaluation of the patient's baseline medical history and cardiac history. Review of the patients ECG pre and post SAH will allow for additional preparation for possible arrhythmias and changes that may be seen intraoperatively.

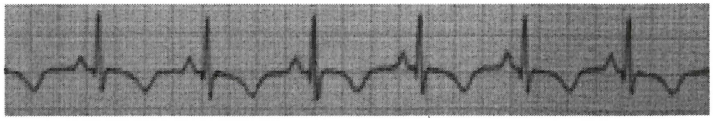
Transthoracic echocardiography can provide valuable information on the patient's systolic function and possible need for inotropic support intraoperatively. Discussion with the neurosurgical team about blood pressure goals and fluid status in context of the patient with ischemic cardiomyopathy help plan an appropriate anesthetic.

Any decision to postpone surgery for further cardiac evaluation must be balanced against the risk of re-bleeding and vasospasm. Most of these patients will not benefit from cardiac interrogation in the Cath lab as the cause of their ischemic changes is not driven by coronary atherosclerosis.

Intraoperatively all of the principles of neuro anesthesia remain unchanged. Invasive arterial monitoring is essential for CO2 management and meticulous blood pressure control. IV access capable of rapid transfusion is necessary to manage possible intraoperative rupture. Patients with cardiac dysrhythmias, in addition to cerebrally induced cardiomyopathies require additional attention to monitor for possible malignant arrhythmias. Vasoactive medications may be required and antiarrhythmic should be available.

Post operatively the use of beta blockade for cardio protection given the catecholamine induced nature of the cardiac dysfunction can be considered. However patients' with SAH may require vasopressors and possible inotropes for hypotension and cardiac failure and beta blockers may need to be withheld.

In summary understanding the pathophysiology of cerebral induced cardiac ischemia in patients with SAH will assist the anesthesiologist in optimal intraoperative care.



References:

Morgan, G. E., Mikhail, M. S., & Murray, M. J. (2006). Clinical anesthesiology. New York: Lange Medical Books/McGraw Hill Medical Pub. Division

Guy, John et al. "Perioperative Management Of Aneurysmal Subarachnoid Hemorrhage". *Anesthesia & Analgesia* 81.5 (1995): 1060-1072.

Suarez JI, Tarr RW, Selman WR. Aneurysmal subarachnoid hemorrhage. N Engl J Med 2006; 354:387.

Wijdicks EF, Kallmes DF, Manno EM, et al. Subarachnoid hemorrhage: neurointensive care and aneurysm repair. Mayo Clin Proc 2005; 80:550.

van der Bilt IA, Hasan D, Vandertop WP, et al. Impact of cardiac complications on outcome after aneurysmal subarachnoid hemorrhage: a meta-analysis. Neurology 2009; 72:635.

Hravnak M, Frangiskakis JM, Crago EA, et al. Elevated cardiac troponin I and relationship to persistence of electrocardiographic and echocardiographic abnormalities after aneurysmal subarachnoid hemorrhage. Stroke 2009; 40:3478.

Junttila E, Vaara M, Koskenkari J, et al. Repolarization abnormalities in patients with subarachnoid and intracerebral hemorrhage: predisposing factors and association with outcome. Anesth Analg 2013; 116:190.